Does perinatal exposure to background levels of dioxins have a lasting effect on the human dentition?

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Introduction

Dioxins and Furans (henceforth jointly referred to as dioxins) are among the most toxic substances known to man, recently having been associated even with malignancy, possible congenital malformations and dental abnormalities (1, 2, 3). Dioxins are poorly degradable in nature, and hence conglomerate in the environment. Dioxins are formed as waste products of combustion processes and thus municipal incinerators are amongst the primary sources of these compounds in The Netherlands.

These polychlorinated aromatic compounds are highly lipophilic, readily pass the placenta, and are then primarily stored in fetal adipose tissues. Furthermore, Olie and van den Berg (1985) showed the presence of relatively high concentrations of dioxins and related substances in the breastmilk of lactating Dutch mothers (4). Thus, unborn children and children fed on human breastmilk are being exposed to relatively high concentrations of dioxins and related substances.

A Finnish study showed that breastfed children exposed to higher dioxin concentrations, via their mother's milk, exhibited more mineralisation defects in their permanent first molars than their lower exposure counterparts (3). In our ongoing study of the development of children with known perinatal exposure to dioxins, we therefore also examined the dental statuses of our study participants. We then tested our hypothesis that the higher exposure group would exhibit more dental abnormalities of the permanent first molars than the lower exposure group.

Subjects, Materials and Methods

The study was performed with a group of 41 children aged between 7 and 12 years, with known perinatal dioxin exposure, consenting to a dental examination and light photographs of the dentition.

The children were allocated a time on one of 3 Saturday afternoons during the spring of 1998 for the examination and photographs. The toxic equivalency and cumulative toxic equivalency values as determined by Pluim, et al, were used as such within this follow-up study. Briefly, the concentration of dioxins, using the I-Teq method, in the mother's breastmilk, shortly after having given birth, was taken as the prenatal dioxin exposure level of the child (teqdiox). The postnatal cumulative dioxin exposure (teqcum) was calculated according to the formula: teqdiox multiplied by the amount of breastmilk ingested during the breastfeeding period of the child. We refer you back to Pluim, et al, for a more in-depth explanation of the methods used (5). The prenatal exposure ranged from 8.74 ng/kg fat to 88.8 ng/kg fat with a mean of 34.6 ng/kg fat (N = 41, SD = 18.25). The postnatal exposure ranged from 4.34 ng to 384.51 ng with a mean of 75.4 ng TEQ dioxin (N = 41, SD = 77.54).

Dental abnormalities

The dental status of the children was performed by a practising dentist (M.F.G.) and the light photographs of the dentition were taken by an experienced hospital-photographer. Based upon the examination and photographs, the dentist, who remained blinded to the perinatal dioxin exposure until she had completed the evaluations and analysis, classified the particular child's permanent first molar status using a value from 1 to 4. These numerals were defined as follows:

1 = no visible abnormalities
2 = Caries visible
3 = Enamel abnormalities visible
4 = Caries and enamel abnormalities visible

Statistical analysis

Statistical analysis was performed using the software package SPSS6. A linear regression curve and a bivariate analysis were performed, in order to detect a possible relation between the perinatal dioxin exposure and the current dental status of the study participants. Length of breastfeeding was considered as possible confounder.

Results

Dental abnormalities

25 Children exhibited no dental abnormalities and were assigned a status of 1, 3 children had exclusively caries (status of 2), 7 had exclusively enamel abnormalities (status of 3) and 6 exhibited both caries and enamel abnormalies and were assigned a status of 4 (tables 1 and 2).

Prenatal exposure

No relation was found between the prenatal dioxin exposure and the dental status of the children at the age of 7 – 12 years. The distribution of the dental abnormalities seen showed no obvious trend towards increasing abnormalities with increasing prenatal dioxin exposure.

Postnatal exposure

In contrast to the results of a recent Finnish study (3) we found no obvious relation between the postnatal dioxin exposure and the dental status of our study participants. Furthermore, we found no obvious trend towards increasing dental abnormalities with increasing postnatal dioxin exposure.

Conclusion

In conclusion, while it is quite possible (probable?) that increasing perinatal dioxin exposure leads to increasing dental abnormalities, we have not been able to confirm that this is indeed the case amongst our longitudinal dioxin exposure study participants.

Bibliography

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Tables

	Number of children with prenatal dioxin exposure =< 31 ng/kg fat	Number of children with prenatal dioxin exposure > 31 ng/kg fat
Dental Status	N = 21	N = 20
1 (no visible abnormalities)	14	11
2 (caries)	2	1
3 (enamel abnormalities)	3	4
4 (caries + enamel abnormalities)	2	4

Table 1: Dental status and prenatal dioxin exposure.

	Number of children with postnatal dioxin exposure =< 50 ng/kg fat	Number of children with postnatal dioxin exposure > 50 ng/kg fat
Dental Status	N = 21	N = 20
1 (no visible abnormalities)	12	13
2 (caries)	2	1
3 (enamel abnormalities)	4	3
4 (caries + enamel abnormalities)	3	3

Table 2: Dental status and postnatal dioxin exposure.